HLA DR4 = (DRB1(*)0405)
HLA DR53 = (DRB4(*)0101 - Table 6

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 NEWS  4  Feb 16  TOXLINE no longer being updated
 NEWS  5  Apr 23  Search Derwent WPINDEX by chemical structure
 NEWS  6  Apr 23  PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
 NEWS  7  May 07  DGENE Reload
 NEWS  8  Jun 20  Published patent applications (A1) are now in USPATFULL
 NEWS  9  JUL 13  New SDI alert frequency now available in Derwent's
                  DWPI and DPCI
 NEWS 10  Aug 23  In-process records and more frequent updates now in
                  MEDLINE
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 NEWS 13  Sep 17  IMSworld Pharmaceutical Company Directory name change
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 NEWS 14  Oct 09  Korean abstracts now included in Derwent World Patents
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 NEWS 15  Oct 09  Number of Derwent World Patents Index updates increased
 NEWS 16  Oct 15  Calculated properties now in the REGISTRY/ZREGISTRY File
 NEWS 17  Oct 22  Over 1 million reactions added to CASREACT
 NEWS 18  Oct 22  DGENE GETSIM has been improved
 NEWS 19  Oct 29  AAASD no longer available
 NEWS 20  Nov 19  New Search Capabilities  USPATFULL and USPAT2
 NEWS 21  Nov 19  TOXCENTER(SM) - new toxicology file now available on STN
 NEWS 22  Nov 29  COPPERLIT now available on STN
 NEWS 23  Nov 29  DWPI revisions to NTIS and US Provisional Numbers
 NEWS 24  Nov 30  Files VETU and VETB to have open access
 NEWS 25  Dec 10  WPINDEX/WPIDS/WPIX New and Revised Manual Codes for 2002
 NEWS 26  Dec 10  DGENE BLAST Homology Search
 NEWS 27  Dec 17  WELDASEARCH now available on STN
 NEWS 28  Dec 17  STANDARDS now available on STN
 NEWS 29  Dec 17  New fields for DPCI
 NEWS 30  Dec 19  CAS Roles modified
 NEWS 31  Dec 19  1907-1946 data and page images added to CA and CAplus

 NEWS EXPRESS  August 15 CURRENT WINDOWS VERSION IS V6.0c,
               CURRENT MACINTOSH VERSION IS V6.0 (ENG) AND V6.0J (JP),
               AND CURRENT DISCOVER FILE IS DATED 07 AUGUST 2001
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=> s (motif? or consensus)
L1       263181 (MOTIF? OR CONSENSUS)

=> s  l1 (P) (DR53 or (DRB4 (1N) 0101))
L2           40 L1 (P) (DR53 OR (DRB4 (1N) 0101))

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3            14 DUP REM L2 (26 DUPLICATES REMOVED)

=> dis l3 1-14 ibib abs kwic

L3   ANSWER 1 OF 14  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:        2001:209257  CAPLUS
DOCUMENT NUMBER:         135:343017
TITLE:                   Polymorphism in the Y box regulates cytokine-mediated
                         expression of HLA-DR genes
AUTHOR(S):               Sindwani, S.; Singal, D. P.
CORPORATE SOURCE:        Department of Pathology and Molecular Medicine,
                         McMaster University, Hamilton, ON, Can.
SOURCE:                  Transplant. Proc. (2001), 33(1-2), 487-488
```

CODEN: TRPPA8; ISSN: 0[redacted]1345
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of polymorphisms in the XI and Y box **motifs** in DRB promoters on the level of cytokine-mediated expression of DRB genes were examd. Genomic DNA from DR1-, DR51-, **DR53**-pos. homozygous B lymphoblastoid cell lines was PCR-amplified for promoter regions of DRB1 genes. Results suggest that the polymorphism in X1 box does not affect the cytokine-mediated strength of DRB1 promoters. However, substitution of a single nucleotide in the Y box **motif** has a significant effect on the promoter strength. The Y box with a perfect inverted CCAAT sequence enhances the promoter strength, whereas the Y box with an imperfect inverted CCAAT sequence has an inhibitory effect on cytokine-mediated transcriptional activity of DRB promoters.
REFERENCE COUNT: 4
REFERENCE(S): (1) Qiu, X; HLA 1996, P310
(2) Singal, D; Immunogenetics 1993, V37, P143 CAPLUS
(3) Singal, D; Immunogenetics 1996, V43, P50 CAPLUS
(4) Singal, D; Proceedings 10th International Congress of Immunology 1998
AB The effects of polymorphisms in the XI and Y box **motifs** in DRB promoters on the level of cytokine-mediated expression of DRB genes were examd. Genomic DNA from DR1-, DR51-, **DR53**-pos. homozygous B lymphoblastoid cell lines was PCR-amplified for promoter regions of DRB1 genes. Results suggest that the polymorphism in X1 box does not affect the cytokine-mediated strength of DRB1 promoters. However, substitution of a single nucleotide in the Y box **motif** has a significant effect on the promoter strength. The Y box with a perfect inverted CCAAT sequence enhances the promoter strength, whereas the Y box with an imperfect inverted CCAAT sequence has an inhibitory effect on cytokine-mediated transcriptional activity of DRB promoters.

L3 ANSWER 2 OF 14 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001029254 MEDLINE
DOCUMENT NUMBER: 20504242 PubMed ID: 11050037
TITLE: Fine specificity of T cells reactive to human PDC-E2 163-176 peptide, the immunodominant autoantigen in primary biliary cirrhosis: implications for molecular mimicry and cross-recognition among mitochondrial autoantigens.
AUTHOR: Shigematsu H; Shimoda S; Nakamura M; Matsushita S; Nishimura Y; Sakamoto N; Ichiki Y; Niho Y; Gershwin M E; Ishibashi H
CORPORATE SOURCE: The First Department of Internal Medicine, Faculty of Medicine, Kyushu University, Fukuoka, Japan.
CONTRACT NUMBER: DK39588 (NIDDK)
SOURCE: HEPATOLOGY, (2000 Nov) 32 (5) 901-9.
Journal code: GBZ. ISSN: 0270-9139.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001121
AB The anti-mitochondrial antibody response in primary biliary cirrhosis (PBC) is primarily directed at E2 components of PDC, OGDC, and BCOADC, and E3BP. Previous work has shown that the immunodominant autoreactive T- cell epitope is the PDC-E2 163-176 peptide, restricted by HLA **DR53**. To address molecular mimicry and cross-recognition among mitochondrial autoantigens, we analyzed reactivity, including agonism and antagonism assays, to a series of single amino acid-substituted peptides using cloned T-cell lines in PBC and controls. Interestingly, fine specificities were unique for every single T-cell clone, but the clones could be categorized into two distinct groups based on recognition **motifs** of the T-cell receptor (TCR) ligand: group A (170)ExDK(173) and group B (168)EIExD(172). (170)E is the most critical TCR contact residue for both groups of cloned T-cell lines, whereas (173)K and (168)E are the critical TCR contact residues for group A and group B cloned T-cell lines, respectively. More importantly, some group A-cloned T-cell lines cross-reacted to human E3BP 34-47, human OGDC-E2 100-113, and several peptides derived from various microbial proteins carrying an ExDK **motif**, whereas group B-cloned T-cell lines reacted only to E3BP 34-47 carrying an EIExD **motif**. Furthermore, an RGxG **motif** was exclusively found in the complementarity-determining region (CDR3) of the TCR Vbeta in the group B-cloned T-cell lines, while G, S, and/or R were frequently found in the CDR3 of the TCR Vbeta in the group A-cloned T-cell lines. These data provide a framework for understanding molecular mimicry among mitochondrial antigens.
AB . . . E3BP. Previous work has shown that the immunodominant autoreactive T- cell epitope is the PDC-E2 163-176 peptide, restricted by HLA **DR53**. To address molecular mimicry and cross-recognition among mitochondrial autoantigens, we analyzed reactivity, including agonism and antagonism assays, to a series. . . were unique for every single T-cell clone, but the clones could be categorized into two distinct groups based on recognition **motifs** of the T-cell receptor (TCR) ligand: group A (170)ExDK(173) and group B (168)EIExD(172). (170)E is the most critical TCR contact. . . lines cross-reacted to human E3BP 34-47, human OGDC-E2 100-113, and several peptides derived from various microbial proteins carrying an ExDK **motif**, whereas group B-cloned T-cell lines reacted only to E3BP 34-47 carrying an EIExD **motif**. Furthermore, an RGxG **motif** was exclusively found in the complementarity-determining region (CDR3) of the TCR Vbeta in the group B-cloned T-cell lines, while G,. . .

L3 ANSWER 3 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:48724 CAPLUS
DOCUMENT NUMBER: 133:15971
TITLE: Immune pathophysiology of primary biliary cirrhosis
AUTHOR(S): Ishibashi, Hiromi; Shimoda, Shinji; Shigematsu, Hirohisa; Ichiki, Yasunori; Nakamura, Minoru; Hayashida, Kazuhiro; Gershwin, M. Eric
CORPORATE SOURCE: The First Department of Medicine, Kyushu University, Fukuoka, 812-8582, Japan
SOURCE: Int. Congr. Ser. (1999), 1188(Progress in Hepatology, Volume 5: Liver and Immunology), 105-115
CODEN: EXMDA4; ISSN: 0531-5131
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 43 refs. Primary biliary cirrhosis (PBC) is an autoimmune disease of the liver in which the major pathol. feature is destruction of

intralobular bile ducts. The serol. hallmark of PBC is the presence of antimitochondrial antibodies. The major mitochondrial autoantigens have been identified as the E2 component of pyruvate dehydrogenase complex (PDC-E2). We demonstrated that T cell epitopes were mapped close to the B cell epitopes on PDC-E2 163-176 of the inner lipoyl domain and 36-49 of the outer lipoyl domain in PBC patients with HLA **DR53**. The PDC-E2 163-176 peptide-reactive T cells exist in the liver of PBC patients in approx. 100-fold higher frequency. The peptide-specific T cell reacted with the PDC-E2 whole protein, suggesting that the peptide PDC-E2 163-176 may be expressed on HLA mols. following processed in the cell. Some T cell clones also reacted with a peptide originated from Escherichia coli which possess ExDK sequences, suggesting that the amino acid **motif**, ExDK, in the peptide is essential for the immune reaction. We hypothesize that the infection of pathogens such as Escherichia coli would activate T cells to break tolerance to the self-antigen possessing a mimic sequence, which leads to the development of the pathol. changes in PBC.

REFERENCE COUNT: 43

REFERENCE(S):
(2) Coppel, R; Immunol Rev 1995, V144, P17 CAPLUS
(3) Coppel, R; Proc Natl Acad Sci 1988, V85, P7317 CAPLUS
(5) Fukushima, N; Int Immunol 1995, V7, P1047 CAPLUS
(6) Fussey, S; Proc Natl Acad Sci 1988, V85, P8654 CAPLUS
(7) Fussey, S; Proc Natl Acad Sci 1990, V87, P3987 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB A review with 43 refs. Primary biliary cirrhosis (PBC) is an autoimmune disease of the liver in which the major pathol. feature is destruction of intralobular bile ducts. The serol. hallmark of PBC is the presence of antimitochondrial antibodies. The major mitochondrial autoantigens have been identified as the E2 component of pyruvate dehydrogenase complex (PDC-E2). We demonstrated that T cell epitopes were mapped close to the B cell epitopes on PDC-E2 163-176 of the inner lipoyl domain and 36-49 of the outer lipoyl domain in PBC patients with HLA **DR53**. The PDC-E2 163-176 peptide-reactive T cells exist in the liver of PBC patients in approx. 100-fold higher frequency. The peptide-specific T cell reacted with the PDC-E2 whole protein, suggesting that the peptide PDC-E2 163-176 may be expressed on HLA mols. following processed in the cell. Some T cell clones also reacted with a peptide originated from Escherichia coli which possess ExDK sequences, suggesting that the amino acid **motif**, ExDK, in the peptide is essential for the immune reaction. We hypothesize that the infection of pathogens such as Escherichia coli would activate T cells to break tolerance to the self-antigen possessing a mimic sequence, which leads to the development of the pathol. changes in PBC.

L3 ANSWER 4 OF 14 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1998179137 MEDLINE
DOCUMENT NUMBER: 98179137 PubMed ID: 9510558
TITLE: Tyrosinase epitope recognized by an HLA-DR-restricted T-cell line from a Vogt-Koyanagi-Harada disease patient.
AUTHOR: Kobayashi H; Kokubo T; Takahashi M; Sato K; Miyokawa N; Kimura S; Kinouchi R; Katagiri M
CORPORATE SOURCE: Department of Pathology, Asahikawa Medical College, 4-5-3-11, Nishikagura, Asahikawa, 078, Japan.
SOURCE: IMMUNOGENETICS, (1998 Apr) 47 (5) 398-403. Journal code: GI4. ISSN: 0093-7711.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001214

AB Human T-cell-mediated autoimmune diseases are often genetically linked to particular alleles of HLA class II genes. Vogt-Koyanagi-Harada's (VKH) disease, which is regarded as an autoimmune disorder in multiple organs containing melanocytes, has been found to be associated with HLA-DR4 (DRB1(*)0405) and HLA-**DR53** (**DRB4**(*)**0101**). Tyrosinase is a melanoma antigen (Ag) expressed by normal melanocytes as well as melanoma cells against which responses by autologous T cells have been detected. We established a T-cell line from the peripheral blood of a patient with VKH disease which responded to synthetic peptides corresponding to tyrosinase. The T-cell line was generated which recognized the tyrosinase p188 - 208 peptide when presented by the HLA-DR4 (DRB1(*)0405) molecule on the surface of HLA class II-expressing L-cell transfectants. The minimal antigenic peptide which induced T-cell responses was an 11-amino-acid sequence and located at tyrosinase p193 - 203 (E-I-W-R-D-I-D-F-A-H-E). This peptide contained the DRB1(*)0405-binding peptide **motif** (hydrophobic residues (Y, F, W) at position 1 as an anchor residue, and negatively charged residues (D, E) at position 9), which corresponded to the W at p195 and the D at p203. These observations demonstrate that tyrosinase peptides are immunogenic, and may be a candidate for an autoantigen in VKH disease, suggesting that probing the T-cell responses against synthetic peptides is a productive approach for identifying the autoantigenic peptides associated with autoimmune diseases including VKH disease.

AB . . . regarded as an autoimmune disorder in multiple organs containing melanocytes, has been found to be associated with HLA-DR4 (DRB1(*)0405) and HLA-**DR53** (**DRB4**(*)**0101**). Tyrosinase is a melanoma antigen (Ag) expressed by normal melanocytes as well as melanoma cells against which responses by autologous. . . T-cell responses was an 11-amino-acid sequence and located at tyrosinase p193 - 203 (E-I-W-R-D-I-D-F-A-H-E). This peptide contained the DRB1(*)0405-binding peptide **motif** (hydrophobic residues (Y, F, W) at position 1 as an anchor residue, and negatively charged residues (D, E) at position. . .

L3 ANSWER 5 OF 14 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97447764 MEDLINE
DOCUMENT NUMBER: 97447764 PubMed ID: 9303504
TITLE: Analysis of T-cell receptor beta of the T-cell clones reactive to the human PDC-E2 163-176 peptide in the context of HLA-DR53 in patients with primary biliary cirrhosis.
AUTHOR: Ichiki Y; Shimoda S; Hara H; Shigematsu H; Nakamura M; Hayashida K; Ishibashi H; Niho Y
CORPORATE SOURCE: The First Department of Internal Medicine, Faculty of Medicine, Kyushu University, Fukuoka, Japan.
SOURCE: HEPATOLOGY, (1997 Sep) 26 (3) 728-33. Journal code: GBZ; 8302946. ISSN: 0270-9139.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 19971021
Last Updated on STN: 19971021
Entered Medline: 19971008

AB T-cell-mediated autoimmune mechanisms are considered to be involved in the pathogenesis of primary biliary cirrhosis (PBC). In the previous study, we identified the immunodominant T-cell epitope on the E2 component of pyruvate dehydrogenase complex (PDC-E2) in patients with PBC who have HLA-**DRB4*0101**. In this report, we revealed that the frequency of the T cells reactive to the human PDC-E2 163-176 peptide is significantly increased in the peripheral blood of patients with PBC as compared with healthy subjects. We also confirmed that these T cells were all restricted with HLA-DRB4*01 (**DR53**) by using HLA-DR-transfected L cells. These results together with the evidence that the immunodominant B-cell epitope overlaps with the human T-cell epitope of the PDC-E2 antigen indicate that the T cells reactive to this epitope are closely associated with the pathogenesis of PBC at least in patients who have HLA-**DR53**. Therefore, we analyzed the T-cell receptor (TCR) Vbeta sequence of the five different T-cell clones and the three T-cell clones derived from three patients with PBC and healthy subjects, respectively, which are reactive to the human PDC-E2 163-176 peptide in the context of HLA-**DR53**. The Vbeta- and the Jbeta-gene usages were diverse among the T-cell clones (Vbeta11-Jbeta1.4, Vbeta8-Jbeta1.2, Vbeta12-Jbeta2.1, Vbeta10-Jbeta1.5, and Vbeta20-Jbeta2.1) in patients with PBC. By contrast, in the third complementarity determining region (CDR3), G was frequently found and GXG or GXS **motif** was identified in all T-cell clones. Moreover, RGXG **motif** was found in three clones generated from two patients. In healthy subjects, the Vbeta- and the Jbeta-gene usages were also diverse, and GXG and RGXG **motif** were found. These results indicate that the T cells may recognize the ligand (the human PDC-E2 163-176 peptide/HLA-**DR53** complex) using the limited **motif** in the CDR3 region and that the design of CDR3-specific immunotherapy would be possible using these **motifs**.

AB . . . identified the immunodominant T-cell epitope on the E2 component of pyruvate dehydrogenase complex (PDC-E2) in patients with PBC who have HLA-**DRB4*0101**. In this report, we revealed that the frequency of the T cells reactive to the human PDC-E2 163-176 peptide is. . . patients with PBC as compared with healthy subjects. We also confirmed that these T cells were all restricted with HLA-DRB4*01 (**DR53**) by using HLA-DR-transfected L cells. These results together with the evidence that the immunodominant B-cell epitope overlaps with the human. . . T cells reactive to this epitope are closely associated with the pathogenesis of PBC at least in patients who have HLA-**DR53**. Therefore, we analyzed the T-cell receptor (TCR) Vbeta sequence of the five different T-cell clones and the three T-cell clones. . . patients with PBC and healthy subjects, respectively, which are reactive to the human PDC-E2 163-176 peptide in the context of HLA-**DR53**. The Vbeta- and the Jbeta-gene usages were diverse among the T-cell clones (Vbeta11-Jbeta1.4, Vbeta8-Jbeta1.2, Vbeta12-Jbeta2.1, Vbeta10-Jbeta1.5, and Vbeta20-Jbeta2.1) in patients with PBC. By contrast, in the third complementarity determining region (CDR3), G was frequently found and GXG or GXS **motif** was identified in all T-cell clones. Moreover, RGXG **motif** was found in three clones generated from two patients. In healthy subjects, the Vbeta- and the Jbeta-gene usages were also diverse, and GXG and RGXG **motif** were found. These results indicate that the T cells may recognize the ligand (the human PDC-E2 163-176 peptide/HLA-**DR53** complex) using the limited **motif** in the CDR3 region and that the design of CDR3-specific immunotherapy would be possible using these **motifs**.

L3 ANSWER 6 OF 14 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1998082047 MEDLINE
DOCUMENT NUMBER: 98082047 PubMed ID: 9420477
TITLE: Presence of retroelements reveal the evolutionary history of the human DR haplotypes.
AUTHOR: Svensson A C; Andersson G
CORPORATE SOURCE: Department of Cell Research, Uppsala Genetic Center, Swedish University of Agricultural Sciences, Sweden.
SOURCE: HEREDITAS, (1997) 127 (1-2) 113-24. Ref: 73
Journal code: G6D; 0374654. ISSN: 0018-0661.
PUB. COUNTRY: Sweden
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980226
Last Updated on STN: 19990129
Entered Medline: 19980217

AB Comparison of intron sequences has been a successful tool for drawing major conclusions about the evolutionary relationship of DRB genes. This complex family of genes is discussed in this review as well as a proposed model for the evolution of HLA-DR haplotypes. The model is based both on phylogenetic analysis of intron sequences as well as presence of ERV9 LTR elements located at identical position in intron 5 of a number of DRB genes. According to this model, two main evolutionary branches of DR haplotypes exist. The **DR53** haplotype represents one branch, and the second branch contains the DR51, DR52, DR1, and DR8 haplotypes. After the divergence of the **DR53** haplotype, an ERV9 LTR element was inserted in a primordial gene. Consequently, all DRB1 genes as well as the DRB3 gene within haplotypes of the second branch, contain this LTR element. In addition, conserved regulatory sequence **motifs** are found present within these LTR elements that might regulate DRB gene expression. Novel haplotypes are generated by recombinations and the maintenance of the DR haplotype variation as well as the frequent genetic rearrangements observed might be evolutionary advantageous.

AB . . . 5 of a number of DRB genes. According to this model, two main evolutionary branches of DR haplotypes exist. The **DR53** haplotype represents one branch, and the second branch contains the DR51, DR52, DR1, and DR8 haplotypes. After the divergence of the **DR53** haplotype, an ERV9 LTR element was inserted in a primordial gene. Consequently, all DRB1 genes as well as the DRB3 gene within haplotypes of the second branch, contain this LTR element. In addition, conserved regulatory sequence **motifs** are found present within these LTR elements that might regulate DRB gene expression. Novel haplotypes are generated by recombinations and. . .

L3 ANSWER 7 OF 14 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 96376532 MEDLINE
DOCUMENT NUMBER: 96376532 PubMed ID: 8781122

TITLE: Analysis of anchor residue a naturally processed HLA-DR53 ligand.
AUTHOR: Kobayashi H; Kokubo T; Abe Y; Sato K; Kimura S; Miyokawa N; Katagiri M
CORPORATE SOURCE: Department of Pathology, Asahikawa Medical College, Nishikagura 4-5-3-11, Asahikawa 078, Japan.
SOURCE: IMMUNOGENETICS, (1996) 44 (5) 366-71.
Journal code: GI4; 0420404. ISSN: 0093-7711.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199610
ENTRY DATE: Entered STN: 19961025
Last Updated on STN: 19980206
Entered Medline: 19961016

AB The peptide **motif** of the HLA-**DR53** (**DRB4**(*) **0101**) molecule, which is associated with autoimmune diseases including Vogt-Koyanagi-Harada's syndrome, was determined by peptide binding assay using human L plastin p581 - 595 peptide and its substituted analogues. L plastin p581 - 595 peptide is one of the naturally processed peptides bound to HLA-DR9/**DR53** (DRB1(*)0901/**DRB4**(*) **0101**) molecules. The binding affinity of each peptide to the HLA-**DR53** molecule was measured by fluorescence intensity of biotinylated peptides to L cell transfectants expressing HLA-**DR53** molecules, followed by treatment with avidin-fluorescence. Binding of biotinylated peptides to HLA-**DR53** molecules was not inhibited by all single-alanine-substituted nonbiotinylated peptides, indicating that the replaced position was important for binding to the HLA-**DR53** moleule. The inhibitory **motif** is considered to be an HLA-**DR53**-specific binding **motif**, composed of a positively charged residue (K) at position 1, a hydrophobic residue (I) at position 4, positively charged residue (R or K) at position 8 or 9, and another hydrophobic residue (I) at position 10. This predicted **motif** is different from the binding **motifs** of other HLA-DR molecules.

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L3 ANSWER 8 OF 14 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 96128252 MEDLINE
DOCUMENT NUMBER: 96128252 PubMed ID: 8537121
TITLE: Polymorphism in both X and Y box motifs controls level of expression of HLA-DRB1 genes.
AUTHOR: Singal D P; Qiu X
CORPORATE SOURCE: Department of Pathology, McMaster University, Hamilton, Ontario, Canada.
SOURCE: IMMUNOGENETICS, (1996) 43 (1-2) 50-6.
Journal code: GI4; 0420404. ISSN: 0093-7711.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-S80866
ENTRY MONTH: 199602
ENTRY DATE: Entered STN: 19960221
Last Updated on STN: 19980206
Entered Medline: 19960207

AB The HLA class II antigens of the human major histocompatibility complex play an important role in immune response. The quality of the immune response is determined not only by polymorphisms in their coding region, but also by the level of their cell-surface expression which affects, for example, the extent of T-cell activation. We have previously described allelic polymorphisms in the upstream regulatory regions of HLA-DRB genes, which affected DNA-protein interactions and resulted in significantly different promoter strengths. In the present study, we investigated the effect of polymorphisms in the X and Y box **motifs** on the transcriptional activity of DRB1 gene promoters in the DR1, DR51, and **DR53** haplotype groups. We used normal, chimeric, and mutated DRB promoters and compared their relative abilities to initiate transcription of the CAT reporter gene in human B-cell lines. The results show that polymorphisms in both the X1 and Y box **motifs** play a dominant role in the promoter strength. In the gel mobility shift assay, we observed differential ability of nuclear proteins that bind to the polymorphic X1 and Y box elements. The results in the present study confirm earlier data in that the nucleotide variation in the X1 box affects the level of expression of DRB1 genes. In addition, the present data demonstrate that polymorphism in the Y box, which affects the inverted CCAAT sequence, also plays a dominant role in the transcriptional activity of DRB1 promoters.

AB . . . significantly different promoter strengths. In the present study, we investigated the effect of polymorphisms in the X and Y box **motifs** on the transcriptional activity of DRB1 gene promoters in the DR1, DR51, and **DR53** haplotype groups. We used normal, chimeric, and mutated DRB promoters and compared their relative abilities to initiate transcription of the CAT reporter gene in human B-cell lines. The results show that polymorphisms in both the X1 and Y box **motifs** play a dominant role in the promoter strength. In the gel mobility shift assay, we observed differential ability of nuclear. . .

L3 ANSWER 9 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:864584 CAPLUS
DOCUMENT NUMBER: 123:253909
TITLE: Naturally processed HLA-DR9/DR53 (DRB1*0901/DRB4*0101)-bound peptides
AUTHOR(S): Futaki, Gen; Kobayashi, Hiroya; Sato, Keisuke; Taneichi, Maiko; Katagiri, Makoto

CORPORATE SOURCE: 2nd Department of Pat[illegible]gy, Asahikawa Medical College, Asahikawa, 078, Japan
SOURCE: Immunogenetics (1995), Volume Date 1995, 42(4), 299-301
CODEN: IMNGBK; ISSN: 0093-7711
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Previously it was shown the HLA-DR9 (DRB1*0901) is pos. assocd. with birch pollen allergy and myasthenia gravis. The elucidation of the characteristics of naturally processed peptides bound to HLA-DR9 may help in understanding the pathogenesis of these diseases. Here, the authors isolated and sequenced peptides from HLA-DR9/DR53 (DRB1*0901/DRB4*0101). Amino acid sequences of 19 peptides, eluted by acetic acid dissocn., were detd. Eleven were consistent with parts of known protein sequences.

IT Histocompatibility antigens
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(HLA-**DR53**; identification and sequence **motif** for peptides binding to)

IT Peptides, biological studies
RL: BPR (Biological process); PRP (Properties); BIOL (Biological study); PROC (Process)
(identification and sequence **motif** for HLA-DR9/**DR53** binding by)

IT Protein sequences
(identification and sequence **motif** for peptides binding to HLA-DR9/**DR53**)

L3 ANSWER 10 OF 14 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 95366068 MEDLINE
DOCUMENT NUMBER: 95366068 PubMed ID: 7638864
TITLE: DR4Dw4/DR53 molecules contain a peptide from the autoantigen calreticulin.
AUTHOR: Verreck F A; Elferink D; Vermeulen C J; Amons R; Breedveld F; de Vries R R; Koning F
CORPORATE SOURCE: Department of Immunohaematology and Bloodbank, University Hospital Leiden, The Netherlands.
SOURCE: TISSUE ANTIGENS, (1995 Apr) 45 (4) 270-5.
Journal code: VSV; 0331072. ISSN: 0001-2815.
PUB. COUNTRY: Denmark
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199509
ENTRY DATE: Entered STN: 19950921
Last Updated on STN: 19950921
Entered Medline: 19950914

AB Rheumatoid arthritis (RA) occurs more frequently in HLA-DR4+ individuals than in those who do not express this MHC class II molecule. Although the role of this genetic factor in the immunopathology of this autoimmune disease is unclear, the association of RA with HLA-DR4 may indicate that DR4 molecules present autoantigen(s) to T cells. Here we report the analysis of naturally processed peptides, eluted from a mixture of HLA-DR4Dw4 (DRB1*0401) and **DR53** (**DRB4*0101**) molecules isolated from an RA patient-derived EBV-transformed B cell line. Several (size variants of) self-peptides originating from the autologous molecules HLA-A2, HLA-Cw9, HLA-B62, HLA-DR4Dw4 and HLA-**DR53**, were identified. We also found a sequence that has no homology to any protein in the SwissProt protein sequence databank, and a peptide identical to an internal fragment of the autoantigen calreticulin. The association of the identified peptides with cells expressing HLA-DR4Dw4/**DR53** was confirmed by peptide binding analysis. In agreement with previously described peptide binding **motifs** for DR4Dw4, most peptides contained an aromatic residue (Phe, Tyr, Trp) at relative position i and a small hydroxyl-containing residue (Ser, Thr) at i + 5. Our findings indicate that in RA patient-derived EBV-transformed B cells DR4Dw4/**DR53** molecules present a peptide from the autoantigen calreticulin. Interestingly, autoantibodies against calreticulin have been found in various rheumatic diseases, including rheumatoid arthritis. Thus, the analysis of HLA class II-bound peptides can lead to the identification of putative T helper epitopes, which might be involved in the immunopathology of autoimmune diseases.

AB . . . to T cells. Here we report the analysis of naturally processed peptides, eluted from a mixture of HLA-DR4Dw4 (DRB1*0401) and **DR53** (**DRB4*0101**) molecules isolated from an RA patient-derived EBV-transformed B cell line. Several (size variants of) self-peptides originating from the autologous molecules HLA-A2, HLA-Cw9, HLA-B62, HLA-DR4Dw4 and HLA-**DR53**, were identified. We also found a sequence that has no homology to any protein in the SwissProt protein sequence databank, . . . a peptide identical to an internal fragment of the autoantigen calreticulin. The association of the identified peptides with cells expressing HLA-DR4Dw4/**DR53** was confirmed by peptide binding analysis. In agreement with previously described peptide binding **motifs** for DR4Dw4, most peptides contained an aromatic residue (Phe, Tyr, Trp) at relative position i and a small hydroxyl-containing residue (Ser, Thr) at i + 5. Our findings indicate that in RA patient-derived EBV-transformed B cells DR4Dw4/**DR53** molecules present a peptide from the autoantigen calreticulin. Interestingly, autoantibodies against calreticulin have been found in various rheumatic diseases, including. . .

L3 ANSWER 11 OF 14 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 95263021 MEDLINE
DOCUMENT NUMBER: 95263021 PubMed ID: 7744365
TITLE: Analysis of naturally processed peptides bound to HLA-DR4, DR53 (DRB1*0405, DRB4*0101).
AUTHOR: Kinouchi R; Katagiri M
CORPORATE SOURCE: Second Department of Pathology, Asahikawa Medical College, Japan.
SOURCE: HOKKAIDO IGAKU ZASSHI. HOKKAIDO JOURNAL OF MEDICAL SCIENCE, (1995 Jan) 70 (1) 175-81.
Journal code: GA9; 17410290R. ISSN: 0367-6102.
PUB. COUNTRY: Japan
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Japanese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199506
ENTRY DATE: Entered STN: 19950621
Last Updated on STN: 19950621
Entered Medline: 19950609

AB We have isolated peptides bound to HLA-DR4, **DR53** molecules obtained from HLA homozygous cell line cells, EBV-Wa (DRB1*0405, **DRB4*0101**), and determined amino acid sequences of the peptides. Amino acid sequences of 19 peptides were obtained and identified

as peptide fragments of known proteins. O[illegible]ose, macrophage migration inhibitory factor (MIF), beta 2 microglobulin (beta 2m), pyruvate kinase M2 (PKM2) and cathepsin C are considered to be endogenously derived, while transferrin and apolipoprotein B-100 are exogenous proteins. Peptides corresponding to each protein have a shared sequence with amino terminal or carboxy terminal protrusions. Based on the core sequences, putative DR4, **DR53**-binding **motifs** were suggested as Y----T/V--D or Y----T--D.

AB We have isolated peptides bound to HLA-DR4, **DR53** molecules obtained from HLA homozygous cell line cells, EBV-Wa (DRB1*0405, **DRB4*0101**), and determined amino acid sequences of the peptides. Amino acid sequences of 19 peptides were obtained and identified as peptide. . . each protein have a shared sequence with amino terminal or carboxy terminal protrusions. Based on the core sequences, putative DR4, **DR53**-binding **motifs** were suggested as Y----T/V--D or Y----T--D.

L3 ANSWER 12 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:122443 CAPLUS
DOCUMENT NUMBER: 124:195778
TITLE: Polymorphism in both X and Y box motifs controls level of expression of HLA-DRB1 genes
AUTHOR(S): Singal, Dharam P.; Qiu, Xiaohong
CORPORATE SOURCE: Department Pathology, McMaster University, Hamilton, ON, L8N 3Z5, Can.
SOURCE: Immunogenetics (1995), Volume Date 1996, 43(1/2), 50-6
CODEN: IMNGBK; ISSN: 0093-7711
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The HLA class II antigens of the human major histocompatibility complex play an important role in immune response. The quality of the immune response is detd. not only by polymorphisms in their coding region, but also by the level of their cell-surface expression which affects, for example, the extent of T-cell activation. We have previously described allelic polymorphisms in the upstream regulatory regions of HLA-DRB genes, which affected DNA-protein interactions and resulted in significantly different promoter strengths. In the present study, we investigated the effect of polymorphisms in the X and Y box **motifs** on the transcriptional activity of DRB1 gene promoters in the DR1, DR51, and **DR53** haplotype groups. We used normal, chimeric, and mutated DRB promoters and compared their relative abilities to initiate transcription of the CAT reporter gene in human B-cell lines. The results show that polymorphisms in both the X1 and Y box **motifs** play a dominant role in the promoter strength. In the gel mobility shift assay, we obsd. differential ability of nuclear proteins that bind to the polymorphic X1 and Y box elements. The results in the present study confirm earlier data in that the nucleotide variation in the X1 box affects the level of expression of DRB1 genes. In addn., the present data demonstrate that polymorphism in the Y box, which affects the inverted CCAAT sequence, also plays a dominant role in the transcriptional activity of DRB1 promoters.

AB The HLA class II antigens of the human major histocompatibility complex play an important role in immune response. The quality of the immune response is detd. not only by polymorphisms in their coding region, but also by the level of their cell-surface expression which affects, for example, the extent of T-cell activation. We have previously described allelic polymorphisms in the upstream regulatory regions of HLA-DRB genes, which affected DNA-protein interactions and resulted in significantly different promoter strengths. In the present study, we investigated the effect of polymorphisms in the X and Y box **motifs** on the transcriptional activity of DRB1 gene promoters in the DR1, DR51, and **DR53** haplotype groups. We used normal, chimeric, and mutated DRB promoters and compared their relative abilities to initiate transcription of the CAT reporter gene in human B-cell lines. The results show that polymorphisms in both the X1 and Y box **motifs** play a dominant role in the promoter strength. In the gel mobility shift assay, we obsd. differential ability of nuclear proteins that bind to the polymorphic X1 and Y box elements. The results in the present study confirm earlier data in that the nucleotide variation in the X1 box affects the level of expression of DRB1 genes. In addn., the present data demonstrate that polymorphism in the Y box, which affects the inverted CCAAT sequence, also plays a dominant role in the transcriptional activity of DRB1 promoters.

L3 ANSWER 13 OF 14 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 95012459 MEDLINE
DOCUMENT NUMBER: 95012459 PubMed ID: 7927542
TITLE: Peptide **motifs** of HLA-DR4/**DR53** (DRB1*0405/**DRB4*0101**) molecules.
AUTHOR: Kinouchi R; Kobayasi H; Sato K; Kimura S; Katagiri M
CORPORATE SOURCE: Asahikawa Medical College, Second Department of Pathology, Japan.
SOURCE: IMMUNOGENETICS, (1994) 40 (5) 376-8.
Journal code: GI4; 0420404. ISSN: 0093-7711.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199411
ENTRY DATE: Entered STN: 19941222
Last Updated on STN: 19941222
Entered Medline: 19941108

TI Peptide **motifs** of HLA-DR4/**DR53** (DRB1*0405/**DRB4*0101**) molecules.

L3 ANSWER 14 OF 14 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 93224768 MEDLINE
DOCUMENT NUMBER: 93224768 PubMed ID: 8468491
TITLE: Comparison of HLA class II genes in Caucasoid, Chinese, and Japanese patients with primary Sjogren's syndrome.
AUTHOR: Kang H I; Fei H M; Saito I; Sawada S; Chen S L; Yi D; Chan E; Peebles C; Bugawan T L; Erlich H A; +
CORPORATE SOURCE: Department of Immunology and Rheumatology, Scripps Research Institute, La Jolla, CA 92037.
CONTRACT NUMBER: M01 RR 00833 (NCRR)
R01 AR33983 (NIAMS)
SOURCE: JOURNAL OF IMMUNOLOGY, (1993 Apr 15) 150 (8 Pt 1) 3615-23.
Journal code: IFB; 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199305
ENTRY DATE: Entered STN: 19930521
Last Updated on STN: 19930521

Entered Medline: 19930512

AB To better define the genetic factors that predispose to primary Sjogren's syndrome (SS), we have used polymerase chain reaction in combination with oligonucleotide probe hybridization and DNA sequencing to analyze HLA-DRB1, -DQA1, -DQB1, and -DPB1 alleles in Caucasoid (California), Japanese (Tokyo), and Chinese (Shanghai and Beijing) SS patients. In comparison to local controls in each region, we found: 1) increased frequency of the predicted haplotype HLA-DRB1*0301-DRB3*0101-DQA1*0501-DQB1*0201 in Caucasoid patients ($p < 0.001$); 2) increased frequency of the predicted haplotype HLA-DRB1*0405-**DRB4*0101**-DQA1*0301-DQB1*0401 in Japanese patients ($p < 0.05$); 3) increased frequency of the predicted haplotype DRB1*0803-DQA1*0103-DQB1*0601 in Chinese patients ($p < 0.05$); and 4) no statistically significant association with DPB1 alleles in any group, although an increased number of Caucasoid and Japanese SS patients possessed DPB1*0301. Comparison of DNA sequences for the three disease-associated haplotypes in these ethnic groups revealed a shared region of predicted amino acids from positions 58 to 69 in the first domain of HLA-DQB1. These results extend previous studies by demonstrating that no single class II allele was associated with 1 degree SS in the different ethnic groups. However, a shared amino acid **motif** in the DQB1 first domain was present in each disease-associated haplotype.

AB . . . increased frequency of the predicted haplotype HLA-DRB1*0301-DRB3*0101-DQA1*0501-DQB1*0201 in Caucasoid patients ($p < 0.001$); 2) increased frequency of the predicted haplotype HLA-DRB1*0405-**DRB4*0101**-DQA1*0301-DQB1*0401 in Japanese patients ($p < 0.05$); 3) increased frequency of the predicted haplotype DRB1*0803-DQA1*0103-DQB1*0601 in Chinese patients ($p < 0.05$);. . . single class II allele was associated with 1 degree SS in the different ethnic groups. However, a shared amino acid **motif** in the DQB1 first domain was present in each disease-associated haplotype.

=> alexander K?/au or Jager E?/au or Chen Y?/au or scanlan M?/au or gure A?/au or Ritter G?/au or old L//AU OR dRIJFHOUT j?/AU
ALEXANDER IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s alexander K?/au or Jager E?/au or Chen Y?/au or scanlan M?/au or gure A?/au or Ritter G?/au or old L//AU OR dRIJFHOUT j?/AU
L4 45334 ALEXANDER K?/AU OR JAGER E?/AU OR CHEN Y?/AU OR SCANLAN M?/AU OR GURE A?/AU OR RITTER G?/AU OR OLD L//AU OR DRIJFHOUT J?/AU

=> s l4 and (DR53 or (DRB4 (1N) 0101))
L5 7 L4 AND (DR53 OR (DRB4 (1N) 0101))

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6 4 DUP REM L5 (3 DUPLICATES REMOVED)

=> dis l6 1-4 ibib abs

L6 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:78635 CAPLUS
DOCUMENT NUMBER: 134:146372
TITLE: Determining antibodies to NY-ESO-1 in cancer patients
INVENTOR(S): **Jager, Elke**; Stockert, Elisabeth; Old, Lloyd J.; Knuth, Alexander; **Chen, Yao-Tseng**; **Scanlan, Matthew**
PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research, USA; Memorial Sloan-Kettering Cancer Center; Cornell Research Foundation
SOURCE: PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 6
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001007917	A1	20010201	WO 2000-US19220	20000714
W: AU, CA, CN, JP, KR				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 6251603	B1	20010626	US 1999-359503	19990723

PRIORITY APPLN. INFO.:
US 1999-359503 A 19990723
US 1996-725182 A2 19961003
US 1997-937263 A2 19970915
US 1998-62422 A2 19980417
US 1998-165546 A2 19981002

AB The invention relates to methods for detg. tumor status by detg. antibodies specific to NY-ESO-1 in patient samples. One can det. whether a cancerous condition is progressing, regressing, or remaining stable by detg. antibodies against NY-ESO-1 in a patient sample, and comparing the value obtained to a prior value. When the tumor in question expresses NY-ESO-1, a change in this value is indicative of a change in status of the cancerous condition. Anti-NY-ESO-1 antibody was detd. in cancer patient sera by ELISA.
REFERENCE COUNT: 6
REFERENCE(S):
(1) Conrad, K; JOURNAL OF AUTOIMMUNITY, 2nd International Congress on Autoimmunity 1999, SUPPL, P42
(2) Jaeger, E; INTERNATIONAL JOURNAL OF CANCER 1999, V84(5), P506
(3) Lethe, B; INTERNATIONAL JOURNAL OF CANCER 1998, V76(6), P903 CAPLUS
(4) Ludwig Inst Cancer Res; WO 9814464 A 1998 CAPLUS
(5) Stockert, E; JOURNAL OF EXPERIMENTAL MEDICINE 1998, V187(8), P1349 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:468183 CAPLUS
DOCUMENT NUMBER: 135:75726
TITLE: Determination of antibodies to NY-ESO-1 for analysis of tumor status
INVENTOR(S): **Jager, Elke**; Stockert, Elisabeth; Old, Lloyd J.; Knuth, Alexander
PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research, USA
SOURCE: U.S., 19 pp., Cont.-in-part of U.S. Ser. No. 165,546.
CODEN: USXXAM
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 6
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6251603	B1	20010626	US 1999-359503	19990723
US 5804381	A	19980908	US 1996-725182	19961003
US 6274145	B1	20010814	US 1997-937263	19970915
US 6252052	B1	20010626	US 1998-62422	19980417
US 6255470	B1	20010703	US 1999-396184	19990914
WO 2001007917	A1	20010201	WO 2000-US19220	20000714

W: AU, CA, CN, JP, KR
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

PRIORITY APPLN. INFO.:
US 1996-725182 A2 19961003
US 1997-937263 A2 19970915
US 1998-62422 A2 19980417
US 1998-165546 A2 19981002
US 1998-13150 A3 19980126
US 1999-359503 A 19990723

AB The authors disclose methods for anal. of tumor status by detg. antibodies specific to NY-ESO-1. The antibodies can be assessed by ELISA or Western blot; the results of which indicate whether a cancerous condition is progressing, regressing, or remaining stable.

REFERENCE COUNT: 3
REFERENCE(S):
(1) Anon; WO 9814464 1998 CAPLUS
(2) Chen; US 5804381 1998 CAPLUS
(3) Lethe; US 5811519 1998 CAPLUS

L6 ANSWER 3 OF 4 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000150116 MEDLINE
DOCUMENT NUMBER: 20150116 PubMed ID: 10684854
TITLE: Identification of NY-ESO-1 epitopes presented by human histocompatibility antigen (HLA)-**DRB4*** **0101**-0103 and recognized by CD4(+) T lymphocytes of patients with NY-ESO-1-expressing melanoma.
AUTHOR: **Jager E**; Jager D; Karbach J; **Chen Y T**; **Ritter G**; Nagata Y; Gnjatic S; Stockert E; Arand M; Old L J; Knuth A
CORPORATE SOURCE: Medizinische Klinik II, Hamatologie-Onkologie, Krankenhaus Nordwest, 60488 Frankfurt, Germany.. 100333.1434@compuserve.com
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (2000 Feb 21) 191 (4) 625-30.
Journal code: I2V; 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000427
Last Updated on STN: 20000427
Entered Medline: 20000419

AB NY-ESO-1 is a member of the cancer-testis family of tumor antigens that elicits strong humoral and cellular immune responses in patients with NY-ESO-1-expressing cancers. Since CD4(+) T lymphocytes play a critical role in generating antigen-specific cytotoxic T lymphocyte and antibody responses, we searched for NY-ESO-1 epitopes presented by histocompatibility leukocyte antigen (HLA) class II molecules. Autologous monocyte-derived dendritic cells of cancer patients were incubated with recombinant NY-ESO-1 protein and used in enzyme-linked immunospot (ELISPOT) assays to detect NY-ESO-1-specific CD4(+) T lymphocyte responses. To identify possible epitopes presented by distinct HLA class II alleles, overlapping 18-mer peptides derived from NY-ESO-1 were synthetized and tested for recognition by CD4(+) T lymphocytes in autologous settings. We identified three NY-ESO-1-derived peptides presented by **DRB4*0101**-0103 and recognized by CD4(+) T lymphocytes of two melanoma patients sharing these HLA class II alleles. Specificity of recognition was confirmed by proliferation assays. The characterization of HLA class II-restricted epitopes will be useful for the assessment of spontaneous and vaccine-induced immune responses of cancer patients against defined tumor antigens. Further, the therapeutic efficacy of active immunization using antigenic HLA class I-restricted peptides may be improved by adding HLA class II-presented epitopes.

L6 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:690967 CAPLUS
DOCUMENT NUMBER: 131:335782
TITLE: Cloning, tissue distribution, and immunol. characterization of NY-ESO-1
INVENTOR(S): Stockert, Elisabeth; **Jager, Elke**; **Chen, Yao-Tseng**; **Scanlan, Matthew**; **Alexander, Knuth**; Old, Lloyd J.; **Gure, Ali**; **Ritter, Gerd**
PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research, USA
SOURCE: PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 6
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9953938	A1	19991028	WO 1999-US6875	19990324
US 6252052	B1	20010626	US 1998-62422	19980417
AU 9933706	A1	19991108	AU 1999-33706	19990324
EP 1071443	A1	20010131	EP 1999-915110	19990324

W: AU, CA, CN, JP, KR, NZ, ZA
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

PRIORITY APPLN. INFO.:
US 1998-62422 A 19980417
US 1998-165546 A 19981002
US 1996-725182 A2 19961003
US 1997-937263 A2 19970915
WO 1999-US6875 W 19990324

AB The authors disclose the sequence characterization of NY-ESO-1, a tumor-assocd. antigen isolated from esophageal carcinoma. The authors provide distribution of NY-ESO-1 in normal and malignant tissue. In addn., the NY-ESO-1 antigen is mapped for epitopes stimulating MHC class

I- and class II-restricted responses in T[redacted]ls. These peptides are useful in different therapeutic and diagnostic contexts.

REFERENCE COUNT: 3

REFERENCE(S):
(1) Chen, Y; Proc Natl Acad Sci USA 1997, V94, P1914 CAPLUS
(2) Jager, E; J Exp Med 1998, V187(2), P265 CAPLUS
(3) Ludwig Institute for Cancer Research; WO 98/14464 A1 1998 CAPLUS

=> end
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:y

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